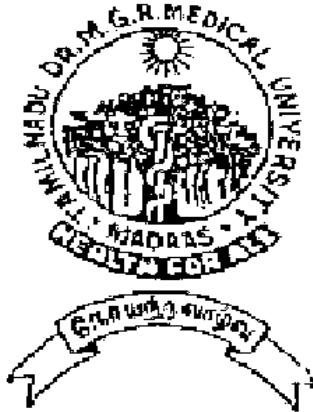


**A STUDY OF CLINICAL PRESENTATION AND
MANAGEMENT OF INTRA ABDOMINAL
HYDATID CYSTS IN GOVT. RAJAJI HOSPITAL,
MADURAI 2006-2008**

**DISSERTATION SUBMITTED FOR
M.S. DEGREE (GENERAL SURGERY)
MARCH – 2009**



**The Tamil Nadu Dr M.G.R. Medical University
Chennai.**

**DEPARTMENT OF SURGERY,
MADURAI MEDICAL COLLEGE AND
GOVERNMENT RAJAJI HOSPITAL,
MADURAI.**

CERTIFICATE

This is to certify that the dissertation entitled “**A STUDY OF CLINICAL PRESENTATION AND MANAGEMENT OF INTRA ABDOMINAL HYDATID CYSTS**” is a bonafide record of work done by **Dr.S.BALAKUMARAN**, in the Department of Surgery, Government Rajaji Hospital, Madurai Medical College, Madurai., under the direct guidance of me.

**Professor of Surgery
and unit chief**

**Professor and HOD
Department of surgery,
Madurai Medical College and
Government Rajaji Hospital,
Madurai.**

**DEAN
Madurai Medical College and
Government Rajaji Hospital
Madurai.**

DECLARATION

I, **Dr. S.BALAKUMARAN**, hereby declare that I carried out this work on, **“A STUDY OF CLINICAL PRESENTATION AND MANAGEMENT OF INTRA ABDOMINAL HYDATID CYSTS”** at the Department of General Surgery, Govt. Rajaji Hospital, Madurai during the period of June 2006 to November 2008. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree or diploma to any other University, Board either in India or abroad.

This is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S degree examination in General Surgery.

Place : Madurai

Dr. S.BALAKUMARAN

Date :

CONTENTS

ACKNOWLEDGMENT

INTRODUCTION	1
1. AIM OF STUDY	3
2. GENERAL ACCOUNT OF THE PARASITE	4
3. MATERIALS AND METHODS	18
4. CLINICAL FEATURES	19
5. TREATMENT MODALITIES	28
6. HEPATIC HYDATID CYSTS	32
7. HYDATID CYSTS IN OTHER SITES	43
8. DISCUSSION	44
9. CONCLUSION	54
BIBLIOGRAPHY	55
MASTER CHART	

ACKNOWLEDGMENT

I wish to thank my Unit chief **Prof.Dr.K.V.MAHESWARAN** who has helped me in compiling this work through thoughtful advice. I also thank my assistant professors for their guidance and help. I also appreciate the contribution made by the Department of SGE. I am also grateful to the patients in the surgical ward without whose help this work would not have been possible.

INTRODUCTION

Echinococcosis or Hydatidosis is a Zoonotic infestation prevalent in most of the countries of the world in varying degrees. In India, it is prevalent widely in parts like Punjab and Tamil Nadu. The causative agents are a few species of the cyclophyllidean cestodes of the genus *Echinococcus*. A survey of the literature shows that there is no precise data on the incidence of hydatid disease in India. Stry reports of human cases have been published from time to time from various parts of the country. Mahadevan and Menon reported from General Hospital Chennai, 8 cases of hydatid disease for the period from 1923 to 1932. Athma Rama Rao (1952) reported one case of hydatid cysts in Kidney. Reddy and Anguli (1953) reported 33 cases for the period from 1933 to 1952. Bhat and Carman (1956) reported 33 cases from CMC Vellore for a period of 20 years. D.J.Reddy et al from Guntur reported 9 cases of which 4 were from the liver. Rao and Balasubramanian (1969) reported 2 cases of hydatid cyst of spleen. Wagle (1964) reported 107 cases for a period of 5 years from 1955. Gupta (1966) from Indore reported 31 cases in 15 years. Reddy and Saradha (1968) published a review of 63 cases from Guntur for a period of 12 years from 1955 to 1956. K.N.Vasudevan (1967) reported 40 cases from Madurai for a period of 14 months from July 1965 to Sept. 1966. Again from Madurai, Srinivasan (1968) reported 60 cases

of pulmonary hydatid cyst. Surya Narayanan from Guntur reported 55 cases of hydatid disease for a period from 1966 to 1969.

It appears from the forgoing account that the infestation is prevalent in widely separated parts of the country. Madurai forms one of the endemic pockets of the disease. The occurrence of the hydatid disease in human beings is fairly high in Madurai. This shows that the infestation by *Echinococcus* in Madurai must be high (i.e.) in cattle, sheep and dog. The dogs gain access easily by eating the raw infected liver, lung or flesh of cattle and the life cycle is continued. It explains the probable cause for it being endemic in and around in Madurai.

AIM OF STUDY

My study is confined to the following aspects of hydatid infestation:

1. Clinical presentation of Intra Abdominal hydatid infestation in man.
2. A critical review of the diagnostic aids of the disease.
3. Methods of treatment of this disease.

GENERAL ACCOUNT OF THE PARASITE

HISTORICAL DATA:

Rudolf (1801) first used the word Echinococcus. The Greek terms 'Echinos' and 'Kokkus' mean spine and berry respectively. The multitude of little cysts resembling berries and hook lets are seen on the scolices within the cyst. The word Hydatid is also of a Greek origin meaning a drop of water.

Hippocrates and Aretoeus and Galen were all clinically familiar with hydatid disease. Reddy (1684), P.J.Hartman (1685) and Tyson (1691) first suspected their animal nature. Palls (1766) first mentioned the similarity of hydatid in man and other mammals. Goeze first studied the scolices of the larvae and recognized their relationship to those of Taenia.

Batsch (1786) recognized under microscope the small Taenia armed with a crown of hook lets in the cyst fluid. The name Taenia-visceralis, scoliosis granulose was given and later Echinococcus granulosus. P.J.Hartman and later Rudolf first studied the adult worm in the dogs intestine. In 1821 Bremser from Vienna reported the first case of hydatid disease in a human patient who was operated upon by Kern.

Von Siebold (1852) followed by Haubner, Leuckart, and Nettleship fed scolices of cysts from animals to dogs and developed the adult worm in the host's intestine. Virchow described the alveolar form of this disease in man. Leuckart (1863) suggested the name *Echinococcus multilocularis* for the parasite which produced alveolar hydatid disease. Nannya in Germany, Finsen in Iceland and Thomas in Australia, bred adult worms in dogs from scolices of human origin. Dave and Sri Harold Dew studied the problem extensively particularly the evolution of hydatid cyst and there from the basis of modern development on this subject.

N.R.Barrett (1949) devised a technique for removal of a solitary univesicular pulmonary hydatid cyst. Again N.R.Barrett and D.Thomas have written extensively on the pathological status of the pulmonary hydatid disease. J.Barrie (1965) experimentally proved the potential risk of pulmonary hydatid disease from inhalation of *Echinococcus* ova.

GEOGRAPHICAL DISTRIBUTION:

The parasite is cosmopolitan in distribution and therefore hydatid disease is universal. It tends to become a public health to man when the ratio of sheep to man in a country exceeds 0.4:1. With the intensification of agriculture in a hungry world this disorder cannot be neglected.

Hydatid disease in man was common enough in Europe at the beginning of this century, but in recent years it is assumed that the disease occurs only with any frequency in the more temperate zones of the Southern Hemisphere. According to Dew the classical hydatid countries are Iceland, Australia, New Zealand and South America.

The most extensive and intensive enzootic regions are as follows:

1. South Australia, Tasmania and New Zealand.
2. Southern half of South America including Argentina, Paraguay, Chile, Uruguay and Southern Brazil.
3. Northern and Southern part of Africa.
4. Northern Southern and Eastern Europe, Siberia, Turkey and Greece.
5. Northern China, Mongolia and Philipines.

Hydatidosis occurs principally in the cattle raising countries of the world, since there occur a close relation between ungulates and dogs. In these countries hydatid disease is a major cause of morbidity and mortality. In Iraq it is the most commonly encountered surgical problem. In Uruguay a major medical journal is concerned exclusively with hydatidosis. High incidence rates are reported from Argentina Chile and Peru. Thus infection seems to be more prevalent in the temperate regions

of the world. Australia and New Zealand have remained major endemic areas.

In India the disease is endemic in Punjab and certain parts of South India, Andhra Pradesh and Southern parts of Tamil Nadu. In many endemic areas of hydatid disease dogs are used as herd animals. In Lebanon dog faces were used for tanning leather and therefore shoemakers were often infected. In Turkana and Kenya dogs are used as nurses to guard children and to clean them when they vomit or defecate. The high incidence in Turkana is compounded by the unusual circumstance of man acting as an intermediate host because with the absence of burial customs dogs and jackals have an easy access to human corpses.

Muslims tend to avoid direct contact with dogs because Prophet Mohammed had warned that “If a dog enters your house an angel will not enter and if a dog licks your kitchen utensils it must be washed seven times”. Even then it is common in certain Muslim countries because of the poor disposal of the infected offal, particularly after religious feasts when almost every family slaughters a sheep and the surplus meat is eaten by dogs and thus the cycle continues.

Table No.: 1 (Comparison of present series to previous series)

Organs Affected	Barrett's 1943	Reddy & Anguli 1953	Gupta 1966	Present 2008
Total No. of Cases	1732	33	31	30
1. Liver	66%	36.4%	22.6%	60%
2. Lung	23%	3%	48.4%	26.6%
3. Peritoneum, Omentum	-	12%	-	3.3%
4. Spleen	1%	-	3.2%	6.6%
5. Kidney	2%	-	3.2%	3.3%
6. Eye	-	3%	9.6%	-
7. Bone	2.1%	-	-	-
8. CNS	0.5%	-	-	-
9. Muscle	4%	18.1%	-	-
10. Breast	-	6%	-	3.3%
11. Retro peritoneum	-	-	-	3.3%

SYSTEMATIC POSITION:

Phylum : Helminthes

Subphylum : Platy helminthes

Class : Cestidea

Subclass : Cestoda

Order : Cyclophyllidate

Family : Taenidae

Genus : Echinococcus

Species of Echinococcus:

Recent revision of the genus Echinococcus places within it four species:

1. E.Granulosus
2. E.Multilocularis
3. E.Oligarthrus
4. E.Pantagonicus

CHARACTERISTICS OF ECHINOCOCCUS:

1. E.Granulosus:

This species of Echinococcus is adapted to the host having a well defined predator-prey relationship. At higher latitudes when E.Granulosus exists under natural conditions the final host is the wolf and larval cestode occurs in various species of wild animals upon the wolves regularly prey.

Wild herbivores that become domesticated are also involved in the life cycle of E.Granulosus. Through the domestication of various animals a synanthropic cycle becomes established with the domestic dog replacing the wolf. As a result of introduction of domestic animals from Europe to other parts of the world E.Granulosus has become cosmopolitan in distribution.

The adult worm is not host specific because it has been recorded from various wild carnivores. The larvae of *E. Granulosus* adapted to development in comparatively long lived intermediate host grow relatively slowly.

2. *E. Multilocularis*:

This appears to have a limited distribution in the Northern Hemisphere. The strobilar stage of *E. Multilocularis* appears to be much more host specific. The domestic dog may be the final host for both species and both can develop synchronously in a single dog. Microtine rodents and other mammalian prey of foxes serve as the intermediate host *E. Multilocularis*. These animals are of short life span usually not more than a year. So the larval *E. Multilocularis* develop rapidly reaching a maximum production of protoscolices within a few months.

3. *E. Oligarthrus*:

It appears to be distributed in a restricted way to Central and Southern America a specific distinction of *E. Oligarthrus* has been confirmed by Thatcher and Susan. Some evidence exists to indicate that the larval stage can develop in man.

4. E.Pantagonicus:

It is known only from wild canines in Pantagonis. The natural intermediate host is not known. Szidat (1963) suggested that the larva may produce in man as alveolar cyst formation similar to that caused by E.Multilocularis.

Biology of the Parasite:

1) Habitat:

Adult Echinococcus is parasitic in the villi of the small intestine of carnivores like dog, jackal, fox and wolf. Dog is the preferential definite host. The larval stage (hydatid cyst) occurs in cattle and sheep.

2) Morphology:

The adult worm is one of the smallest tape worms of 3mm to 6mm in length. The body consists of a pyriform scolex 300 μ in diameter and an attenuated neck and strobila made up of one immature, one mature and one gravid proglottid. The terminal proglottid is the largest and longest measuring 2mm to 3mm long and 0.6mm broad. Testes are numerous spherical bodies and the ovary is tubular containing about 500 to 800 eggs. The scolex at its vertex has a rostellum around which is a double row of hooklets 28 to 40 in number and behind them are the few suckers.

3) Life Cycle:

Eggs are ovoid 32-36 μ in length and 25-30 μ in breadth and contain a hexacanth embryo. The eggs are resistant to moisture, extremes of temperature and light and may remain viable for many years. They are passed in the faeces of the definite host which when ingested by a suitable intermediate host such as cattle, sheep, pig or man, the hexacanth embryo is rapidly hatched in the stomach or upper small intestine by dissolution or disruption of its chitinous shell and becomes very active, passing through the bowel wall making its way into a tributary of portal vein and from there being carried to the capillary bed of the liver (1st filter). Bile is known to be involved in the hatching and activation of these cestode eggs. Deveder suggested that the embryo may enter the lacteals and is carried to the venous circulation through the thoracic duct and may get lodged in the lung. In some cases the embryo retains considerable active amoeboid motility and it could squeeze through the liver capillaries resulting in the infestation of the lungs. Subsequently penetration through the pulmonary capillary bed is also possible so that peripheral implantation in various other organs of the body can occur. The ova excreted by the dogs may also enter the lungs directly by inhalation.

On settling down the embryo develops into a hydatid cyst. Inside the cyst innumerable scolices develop. The life cycle is completed when; infected viscera containing the larval stage are eaten by the definite hosts. The scolices get attached to the mucous membrane of the small intestine of the definite host and develop into an adult tapeworm in 6 to 9 weeks.

There is a sylvatic cycle existing in Alaska and Canada involving carnivores such as wolves, jackals and the intermediate hosts being moose and reindeer. It is believed that man is an accidental intermediate host of *Echinococcus*.

4) Development of Hydatid Cycle:

The embryo once lodged in the organ is transferred into a tiny cyst. The hydatid follicle may be recognized in the liver within 12 hours of infestation as an accumulation of mononuclear cells. Within 79hrs a distinctly eosinophilic reaction is apparent around an enlarged parasite. By the fourth day a cystic cavity is formed. At the end of the seventh day the parasite is more distinctly demarcated from the surrounding liver cells and the vascular reaction is more intense. It would seem that upto this stage the parasite may be overwhelmed and destroyed and that phagocytosis of the embryo may occur. By three weeks the follicle is

much larger (250 μ in diameter) and containing a cyst which is visible to the naked eye. The cyst now possesses a nucleated living inner germinal and an outer laminated layer and a commencement of a fibrous adventitia. Some of the neighboring tissue cells show signs of pressure necrosis. Growth is now usually even and a spherical shape is the rule. At three months elasticity appears in its laminated layer. The outer layers are fibrosing and the adventitia is blended with the surrounding tissue. By the end of five months the wall gets differentiated into:

- 1) Inner nucleated germinal layer (endocyst)
- 2) Outer friable laminated milky opaque non-nucleated layer (ectocyst)
- 3) The adventitious host tissue surrounding the ectocyst is termed as the pericyst. This layer is absent in extra hepatic cysts.

Masses of cells develop from the germinal layer. The cells get vacuolated, stalked and project into the cavity. They are the brood capsules which remain attached or move freely in the fluid. From the inner wall of these capsules scolices develop which invaginate into their own bodies to protect their rostellar hooks from injury. The scolices and brood capsules are collectively called as hydatid sand. In a fully developed cyst the germinal layer is about 10-25 μ in thickness. The

germinal layer is attached very lightly to the laminated membrane. The germinal layer gives rise to the brood capsules and laminated membrane and also secretes the hydatid fluid. Some cysts never produce any brood capsules.

Others become sterile by bacterial invasion or calcification. In a few other cysts scolices are never formed. Such cysts are called sterile cysts or acephalocysts. Another characteristic feature of the acephalocyst is the poor development of the laminated membrane. Incidence of sterile cysts seems to be high being 90% in cattle, 20% in pig and 8% in sheep.

Daughter Cyst Formation:

Endogenous daughter cyst formation occurs commonly in the mother hydatid cyst in man. The daughter cysts are a true replica of the mother cyst, being composed of an outer laminated and inner nuclear layer and usually containing brood capsules and scolices. Daughter cyst formation is regarded as a defense reaction to some noxious stimulus. A fully developed scolex is an end product but the scolices could revert in their life cycle and it seems contrary to all the laws of development of the cestodes. Such retrogressive metamorphosis is not only possible but relatively common and is of great clinical importance.

Hydatid Fluid:

The fluid inside the cyst is accumulated in large quantities and is retained under fairly high tension. It acts a protective buffer and a nutritive medium to the developing scolex. The fluid is colorless and crystal clear having a low specific gravity ranging from 1005 to 1010 with a pH of 6.7. The fluid is not coagulated by heat or acids. It contains Sodium chloride, Sodium Sulphate, Sodium Phosphate, Calcium Salts, Glucose, Creatinine and Inositol. The fluid when absorbed gives rise to allergic manifestations.

Cyst Wall:

The organic and inorganic constituents of the laminated membrane of the cyst wall are as follows:

Proteins	:	7.5%
Carbohydrates	:	2%
Lipids	:	1.3%
Ash	:	1.6%
Water content	:	82.4%

ALVEOLAR CYSTS (MULTILOCLAR CYSTS)

E.Multilocularis has been described for over a century. It is without an insulating capsule and has a capacity for exogenous budding with quiet extrusion into neighboring tissues to form vesicles. These vesicles are filled with a transparent gelatinous substances instead of fluid. Dew (1957) considered it as a pleomorphic variant of E.Granulosus. However in a recent revision of genus Echinococcus, E.Multilocularis is shown as a separate species having distinct morphological and biological characteristics.

MATERIALS AND METHODS

The study of these cases of abdominal hydatid cysts is based on cases of hydatid cyst admitted in Govt. Rajaji Hospital during the period from 2006 to 2008. Various methods of surgical treatment have been adopted. During this period 9 cases of extra Abdominal hydatid cysts have also been recorded.

Though the number of cases is limited an attempt has been made to find out the various presentation of hydatid like in liver, spleen, muscle, peritoneum, and mesentery. Assessment was made regarding the incidence of hydatid relative to age, sex, ESR, Blood count, etc.

USG& CT, are used as diagnostic modalities. Standard surgical procedures were done and postoperative follow-up was for limited period.

CLINICAL FEATURES

HUMAN INFESTATION:

Man, an accidental intermediate host of Echinococcus may be infected in a variety of ways such as:

- 1) Consumption of raw vegetables and drinking of water which is contaminated with excreta of infected definite host.
- 2) Ovo-cutaneous contacts of infected pet animals.
- 3) Inhalation of ova.
- 4) Transmission through flies, beetles, and fomites.

DISTRIBUTION OF CYST IN THE HUMAN BODY:

The majority of hydatid cysts in man are found in the liver, this organ receiving the embryo from the portal area first and acting as the primary filter. Sometimes the parasites pass through the lungs where they get lodged and the remainder making their way to the other parts of the body. They may occur in any region of the body and the lessening proportional incidents as the periphery of the body is reached indicating that distribution by blood stream is the only rational explanation.

In the present series of intra abdominal hydatid disease infection is more common in liver.

CLINICAL MANIFESTATIONS:

1. Latency:

The outstanding feature of a simple uncomplicated cyst is its extraordinary latency. Most hydatid cysts in man are acquired in childhood. Frequently the unilocular cyst grows for a period of 5 to 20 years before it is diagnosed.

2. Presence of Swellings:

The presenting symptom of a hydatid cyst situated in the superficial part of the body is a painless swelling. The cystic mass slowly increases in size.

3. Pressure Effects:

Sooner or later pressure effects may appear and bizarre symptoms may result from direct pressure or distortion of the neighboring viscera. Less frequently the young cysts may lodge in the brain or on a heart valve and may produce a dangerous obstruction, which may cause sudden death of the patient. Its development within osseous tissue may produce rapid erosion of the bone, with multiple fractures and crumbling of the bony structures. In the liver the cyst may occupy the greater part of the right lobe with displacement of the left lobe leading to distortion of the costal

margin or building of the chest or abdominal wall. Hydatid cyst of the lung is invariably intracapsular. The hydatid of the spinal column will produce cord compression leading to paraplegia and other neurological problems.

4. Infection:

Once the hydatid is infected it acts as a chronic infective focus. The patient may have intermittent fever with abdominal pain in cases of infected hepatic hydatid cyst.

5. Allergic Manifestation:

The patient may get urticarial rashes due to slow leak of the cyst wall or may get an anaphylactic shock due to the absorption of the specific protein of the hydatid fluid in a sensitized patient.

COURSE OF THE DISEASE:

1. Death of the Parasite:

The death of the parasite may take place at any time as a result of leakage from the cyst, by the entry of bile or other secretions, from bacterial infection or from senility. With increasing age the adventitia becomes denser, less vascular and undergoes calcification so that

interference with nutritional or osmotic process occurs. After the death of the parasite, the laminated membrane becomes more permeable so that fluid is absorbed, the cyst collapses and caseous degeneration of the parasitic tissue occurs. It is similar to that seen in a tuberculous lesion calcium salts get deposited and the whole cyst wall may get impregnated.

2. Rupture:

As the cyst enlarges it comes into intimate relationship with natural channels such as Bronchi, Bile duct or hollow viscus.

At the point of contact the cyst wall weakens and rupture of the cyst may occur into:

- I. Pleural cavity causing hydropneumothorax, pyopneumothorax, and empyema.
- II. The peritoneal cavity causing local or general peritonitis, choleperitonitis and secondary hydatidosis.
- III. Biliary or renal tracts resulting in severe and typical colic with partial or complete obstruction.
- IV. A large channel such as bronchus resulting in more or less complete emptying of the fluid and the laminated membrane leading to spontaneous cure.

- V. A blood vessel causing severe anaphylaxis or metastatic secondary hydatidosis.

3. Suppuration:

The laminated membrane is a potent antibacterial barrier and the leakage of the cyst is an essential preliminary to suppuration. Infection may be haematogenous or more commonly retrograde from bile duct or bronchus. With established infection the cyst contents tend to be liquefied and discharged.

INVESTIGATIONS:

1. Eosinophilia:

The eosinophilia test, a diagnostic test of longstanding has been shown to be a very insensitive method for the diagnosis of the hydatid disease. Generalized eosinophilia is present in 20-25% of the cases. This sign is suggestive along with other clinical evidence, but it is not pathognomonic. In the present series 85% showed an increased eosinophilic count. In hydatid cyst of the liver ESR is elevated in 35% cases recorded in this series.

2. Presence of Hydatid Material in Motion, Urine and Sputum:

The tough laminated membrane is occasionally found in motion in some cases, in sputum in pulmonary hydatid cyst and in urine in renal hydatid cyst. This is due to the rupture of the cyst. The presence of such material is diagnostic.

3. Casoni's Intradermal Test:

The intradermal test for hydatid disease was introduced by Casoni and has been extensively employed in all parts of the world. Injection of 0.2cc of sterile and un-preserved hydatid fluid into the skin produces a wheal upto 5cm in diameter with pseudopodia in about 20mms. The skin test remains positive for life. The sensitivity of the intradermal test is good in proven cases and rates of between 80-95% of cases have been reported. In this series Casoni's test was not done in any case.

4. Serological tests for Hydatid Disease:

The serological tests have been proved to be useful in the presumptive diagnosis of hydatid disease. The serological methods are as follows:

- a) Compliment fixation test
- b) Haemagglutination test
- c) Bentonite flocculation test

- d) Latex agglutination test
- e) Indirect Fluorescent antibody method
- f) ELISA test

Haemagglutination test:

This is a test of choice today for the diagnosis of hydatid disease.

This test has proved slightly more sensitive than all the other tests.

5. Roentgenographic examination:

- a) X-ray chest: X-ray chest is frequently helpful in the diagnosis of hydatid cyst of the lung and thoracic involvement. Tomogram and Bronchogram may also be done whenever necessary. Mass miniature radiography is necessary in endemic areas in order to detect the asymptomatic cases.
- b) Plane X-ray Abdomen erect posture, cholangiography, barium meal series, portovenogram are useful in peritoneal and hepatic hydatid diseases.

6. Diagnostic Cyst Puncture:

This is an accurate method but it is dangerous due to the likelihood of anaphylactic shock resulting from the escape of hydatid fluid. This test should not be attempted unless the surgeon is immediately prepared to perform a radical operation for removal of the cyst.

7. Ultrasonogram:

Ultrasound scan showing a multivesicular cyst (Cartwheel sign) is pathognomonic. The split wall sign is also diagnostic and is probably a consequence of a fall in cyst pressure, which results in separation of endow and ecto cysts.

GHARBI et al Classification:

- Type I - Pure fluid collection
- Type II - Fluid collection with a split wall (Floating membrane)
- Type III - Fluid collection with Septa (Honey comb image)
- Type IV - Heterogenous echo graphic patterns
- Type V - Reflecting thick walls

8. CT Scan:

This is used in detecting hydatidosis of brain and spinal cord. It is superior in the detection of extra-hepatic cysts. The typical findings on CT range from spherical areas of decreased attenuation to a diagnostic picture of multiple daughter cysts.

Hydatid Thrill:

Hydatid thrill was discovered by Blatin in 1801, it is learnt that the genesis of Hydatid thrill is not due to the daughter cysts but due to the mother cyst itself. It is not appropriate to call the persistence of the thrill as 'after thrill'. The so-called hydatid thrill can also be obtained in other

cysts. Therefore it is not a pathognomonic sign of hydatid cyst. Hence a hydatid thrill may be rightly called as a cystic thrill. In the present series this test was negative in all the cases.

9. MRI:

Provides structural details of hydatid cyst, but adds little more than USG or CT and expensive.

10. ERCP (Endoscopic Retrograde Cholangio Pancreatography):

It may show communication between cyst and Bile ducts and can be used to drain the biliary tree before surgery. The routine use of ERCP is advocated by some to completely define the Bileduct anatomy and to visualize any clinically silent communication between the Bile ducts and cysts.

TREATMENT MODALITIES

1. Prophylaxis

- a) Since the human infection results from handling of the infected dogs or ingesting eggs of the parasite on the raw vegetables or from contaminated clothing strict personal hygiene is recommended particularly a thorough cleaning of the hands is advised before eating.
- b) Dogs should be washed regularly with a disinfectant soap.

- c) Dogs should be prevented from entering the kitchen.
- d) Since the only food readily and economically available to dogs was sheep carcass, dogs should be prevented from eating the carcass in endemic areas.
- e) De-worming of dogs should be carried every 3-4 months. Arecoline hydrobromide in a dose rate of 0.4mg per kg body weight was effective in removing the parasite in 95% of dogs. Treatment of dogs each month with bunamidine hydrochloride may reduce the prevalence rate of hydatidosis in domestic livestock more quickly. It is important to limit the home slaughtering of sheep for human consumption in order to lessen the chances of hydatid disease in dogs. It is necessary to have dog proof sheep killing facilities and offal disposal facilities to reduce the incidence of infestation in dogs.

2. Chemotherapy:

Successful medical treatment of hydatid disease with Mebendazole was proved in 1977, though subsequent studies were less conclusive. Albendazole is similar to mebendazole but has a better absorption profile and its principle metabolite albendazole sulphoxide is an effective scolicide. It is more effective than mebendazole. There is now considerable experience of albendazole in hydatid disease. Recommended

dosage for adults is 400mg twice daily for a period of 28 days followed by 14 drug-free day's upto three cycles. Chemotherapy has been used with some success as adjuvant treatment for pre-operative sterilization of the cyst and post-operatively to protect against dissemination for recurrent and inoperable diseases. Albendazole toxicity includes abnormalities in liver function, which are reversible. In a WHO study two cases of anaphylactic shock related to rupture of cyst following mebendazole therapy has been recorded. It was concluded that medical therapy should be reserved for inoperable cases. Praziquantel, is an effective scolical agent but it is effective more on protoscolices than on germinal membrane. Albendazole can cure Hydatid disease. The role of chemotherapy should be restricted to the elderly or unfit patient, disseminated disease and technically inaccessible cysts. Albendazole and praziquantel may also be used in case of spillage during surgery.

3. Immunological Treatment:

Absolute resistance to the larval stage can be acquired and resistance to a number of cestode species can be artificially induced in a number of hosts. Thus research has been directed to the development of vaccines for use in the control program of Echinococcus.

4. Surgical Treatment:

Surgery is the treatment of choice. The aim of surgical treatment should be:

- a) To gain a direct access to the cyst if possible.
- b) To remove the parasite completely
- c) To prevent contamination of the field by active scolices.
- d) To allow for healing of the residual cavity.

The surgical treatments employed are as follows:

- a) PAIR(Percutaneous aspiration, injection, reaspiration)
- b) Excision of the cyst intact
- c) Evacuation of the cyst contents and closure of the adventitial layer.

With or without drain

- d) Drainage of the infected cyst
- e) Marsupialisation

PROGNOSIS:

Natural cure is possible when all the contents of the cyst are evacuated out through the natural channels, like bronchus in pulmonary hydatid cysts. Otherwise a cyst may continue to grow for many years. Secondary infection as a result of a small leak may kill the parasite and the contents of the cyst are either absorbed or calcified. After complete removal of the cyst in a superficial site the prognosis is good.

HEPATIC HYDATID CYST

Pathology

Hydatid cyst expands slowly and asymptotically and tends to be quite large at presentation. Single liver lesion is present in 75% of patients with right and left hepatic lobes affected in 80% and 20% of cases respectively. Despite this singularity half of the lesions contain daughter cysts and are multi locular in nature Hydatid cysts are typically composed 3 layered cyst walls surrounding a fluid cavity.

Pericyst: It is a thin, indistinct, host derived layer of fibrous tissue present in liver and splenic cysts, but absent in cysts of lung and brain. It represents an adventitial reaction to the parasitic infection. The pericyst provides mechanical support to the hydatid cysts and serves as a metabolic interface between host and parasite. As the cyst grows within the liver, Bile ducts and Blood vessels stretch and finally become incorporated within the structure explaining the propensity for biliary and hemorrhagic complications of cyst growth or reaction. With time portions of pericyst may calcify. Complete calcifications of the pericyst interrupts the nutrient and oxygen supply to the parasite, and thus represent the death of the hydatid cyst.

Other 2 layers are derived from parasite itself.

Laminating Membrane: a 1-2mm thick protenaceous layer found abutting the pericyst.

Germinal Membrane: a single cell layer lining inside of the laminated membrane is the location of actual parasites. Hydatid cyst fluid is secreted actively by the infecting parasites. Thus a tense cyst wall indicates a living hydatid cyst.

Hydatid Fluid

- Viable cyst - Thick, clear, alkaline
- Dead cyst /secondary infected -Opaque fluid
- Biliary Communication - Bile Stained
- Erosion of vessels - Hemorrhagic

E.Multilocularis:

- Because of Exogenous vesiculation there is lack of capsule
- Grow in a branching fashion
- Usually found at hilum
- It causes liver pathology by simple space occupying process or by local encasement (or) Invasion of vascular, lymphatic and Biliary structure.

Clinical Presentation:

Hydatid cysts have slow growth rates and rarely cause symptoms. Approximately 75% presents as an asymptomatic abdominal mass (or) a suspicion calcification on a routine chest x-ray. The cyst is usually quite large (>5cm) when symptoms do occur.

Clinical Manifestations of Hydatid Liver Cysts

Asymptomatic	75%
Symptomatic	25%
Abdominal pain	79%
Dyspepsia	50%
Fever with Chills	30%
Jaundice	25%
Arthritis	5%

Signs:

Right upper quadrant mass -70

Right upper quadrant Tenderness – 30%

Laboratory:

Eosinophilia	35%
Bilirubin >2mg	20%
WBC <10,000/mm ³	10%

COMPLICATIONS OF HYDATID CYST IN DECREASING ORDER OF FREQUENCY

Rupture:

Internal

External

Intra Biliary

Intrathoracic

Intraperitoneal

Secondary Bacterial Infection

Anaphylactic Shock

Liver replacement

Internal rupture:

- Occur as a result of trauma (or) pressure necrosis of Bileduct.
- Pericyst separated from laminated membrane and producing multitocular cyst.
- Fluid appears yellowish and morky

External rupture:

Intra Biliary: (Decompression of cyst into the biliary tree) if previously symptomatic, often experiences a transient decrease in pain dyspepsia. However pain returns when cyst contents and fragments lead to obstructive Jaundice, cholangitis (or) secondary infection. In this

recent study one case presented with obstructive jaundice which was treated with choledochotomy & 'T' tube drainage.

Intra Thoracic Rupture:

When a cyst reaching posterior (or) superior liver capsule, erodes through the diaphragm into the pleural cavity. Patient develops right shoulder pain local inflammatory effect within the pleural space allows the cyst to decompress within the lung parenchyma and the patient may develop a hepatobronchial fistula and subsequent Bronchobilia.

Intra Peritoneal Rupture:

Cysts located to the anterior or inferior surface of the liver may rupture into the peritoneal cavity. The resulting implantation and growth of hydatid organisms on and within the peritoneum and omentum is termed abdominal echinococcosis.

Hepatic Replacement:

A cyst can grow large enough to replace the majority of liver parenchyma and causes hepatic failure. However typically the cyst will rupture before this complication develops.

Secondary Infection:

Simple bacterial infections are rare, Instead, they result from a communication between cyst and biliary system. Hyperbilirubinemia and bilestained fluid are characteristic.

Anaphylactic Shock:

It is an acutely life threatening complication. It is due to internal rupture (or) small external leak of hydatid fluid and various protein fragments that reaches blood stream. Sensitisation occurs simply due to repeated exposure. IgE mediated allergic response occurs. Its amenable to treatment with corticosteroids, Antihistamines and epinephrine.

Complications of E.Multilocularis:

- More frequent than E.grannulosus

Severe cholestasis, portal hypertension, GI Haemorrhage, dissemination of the disease to lungs, brain, heart and other organs.

MANAGEMENT OF HEPATIC HYDATID CYST

I. Conservation Management

- Asymptomatic cysts of <4cm
- Located deep in parenchyma

II. Surgical Management

A. Conservative Surgery

- PAIR (Percutaneous aspiration, injection, reaspiration)
- Laparoscopic Evacuation

Indications:

1. GHARBI Type I or II
2. Anterior Cysts
3. Peripheral cysts
4. 1-3 cysts
5. Small Cysts
6. No (or) Minimal calcification
7. Infected cyst meeting above criteria

B. Radical Surgery

1. Open evacuation
2. Pericystectomy
3. Marsupulisation
 - a. GHARBI Type III or IV

- b. Posterior cysts
- c. Central cysts
- d. >3 cysts
- e. Large cysts
- f. Heavy Calcifications
- g. Complicated Cysts
 - i. Infected cysts meeting above criteria
 - ii. Biliary Communication
 - iii. Pulmonary Communication
 - iv. Peritoneal rupture

4. Liver Resection

- a. Multiple cysts with in proximity of blood each other
- b. Cyst in a relatively safe location (II, III segment)

5. Trans plantation

- a. E.Multiocularis leading to fulminant Hepatic failure from
 - i. Sclerosing cholangitis
 - ii. Biliary sclerosis
 - iii. Budd Chiari syndrome

MANAGEMENT OF COMPLICATIONS

Infected Cysts:

- Treated with de-roofing, evacuation, external drainage.
- Infection produces natural cure of the disease as it alter the permeability of membrane and destruction of daughter cysts.

Biliary Communication:

- Preoperative ERCP is gold standard Both in diagnostic and treatment purposes. In our setup ERCP is not available and not done as a routine.
- Spincterotomy and drainage of daughter cysts with excision of liver cyst offers the cure.
- Simple suture legation of the Biliary communication with catgut offers cure.
- Choledochotomy and T tube drainage may be necessary in some cases. In this series one case had Biliary communication & obstructive jaundice.

Anaphylaxis:

As an emergency treated with Antihistaminics, ephinephrine and steroids.

Renal Hydatid (Echinococcosis):

- It is noted in <2% of cases
- Isolated Involvement is rare.
- Symptoms:
 - ❖ Non progressive dull aching abdominal pain
 - ❖ Passing of membranes in urine
- Investigations:
 - ❖ USG
 - ❖ CT with Contrast
 - ❖ Serological tests
 - ❖ IVP – To know the communication with collecting system

Treatment

1. PAIR
2. Total excision
 - ❖ Wedge resection
 - ❖ Partial nephrectomy
 - ❖ Nephrectomy
3. Excision with capitonnage (or) marsupulisation
 - ❖ Albendazole 10mg/kg – 21days / 3 cycles with an intervening gap of 2 weeks.

HYDATID CYSTS IN OTHER SITES

Hydatid cyst in Peritoneum, Omentum and Mesentery:

One case with hydatid cyst in the peritoneum is recorded in this series. This case had multiple hydatid cysts all over the peritoneum, paracolic area, pelvis and mesentery. This patient might have got secondary hydatidosis in peritoneal cavity. The same case had hydatid cyst in the lesser omentum and the same was removed intact.

Hydatid cyst of Spleen:

It is rare occurring in less than 3% of all hydatidcysts. The spleen is enlarged and sometimes a separate swelling in the spleen may be felt. X-ray shows calcification around the cyst margin. In this series two patients presented with a slightly painful swelling in the left hypochondrium moving freely with respiration. Splenectomy was done for both the cases.

Hydatid cyst of Retroperitoneum:

In this series one case of solitary hydatid cyst of left retroperitoneum his recorded, which was successfully excised in toto after taking all precautions.

DISCUSSION

SEX INCIDENCE

TABLE NO 1
TOTAL NUMBER OF CASES 21

SEX	No of cases	Percentage
MALE	12	57.14%
FEMALE	9	42.86%

In this series hydatid cysts slightly more in males than in females

AGE GROUP

Hydatid cyst may occur at any age group. The age incidence of 21 cases of hydatid cysts in this series are given below.

TABLE NO 2

Total no of cases	21
Maximum	65
Minimum	18
Average	39.5

PRESENTING COMPLAINTS

TABLE NO 3

TOTAL NO OF CASES 21

COMPLAINT	NO OF CASES	PERSENTAGE
ASYMTOMATIC	4	19.5%
ABDOMINAL PAIN	10	48.25%
LOSS OF APPETITE	7	33.3%
FEVER	3	14.28%
JAUNDICE	1	4.9%
URINARY SYMPTOMS	0	0%
PULMONARY SYMPTOMS	0	0%

Most of the patients in this series are presenting with right upper quadrant pain, mass, and loss of appetite .one patient presented with obstructive jaundice. In contrast to other studies only few patients are asymptomatic

ESR

TABLE NO 4

Total No. of Cases 21

INCREASED ESR	7	33.3%
NORMAL ESR	14	66.6%

ESR is raised in Small No. of Cases Only. It is raised in multiple liver cysts & extra hepatic cysts

EOSINOPHILIA

TABLE NO 5

Total No. of Cases 21

INCREASED EOSINOPHIL COUNT	18	85.7%
NORMAL COUNT	3	14.3%

In the present series more than 80% of cases show increased eosinophilic count. But this is not pathognomonic.

ORGAN INVOLVEMENT

TABLE NO 6
Total No. of Cases 21

ORGAN	NO OF CASES	PERSENTAGE
LIVER	16	76.18%
SPLEEN	2	9.52%
LIVER&KIDNEY	1	4.76%
RETROPERITONIUM	1	4.76%
LIVER&PERITONEUM	1	4.76%

As in other series liver is the most common organ affected. followed by spleen, kidney, peritoneum and retroperitoneum.

Peritoneal involvement in this series is secondary to liver involvement.

HEPATIC HYDATID

TABLE NO 7
Total No. of cases 18

SINGLE	11	60.6%	MULTIPLE	7
RIGHT	9	49.5%	39.4%	
LEFT	2	11.1%		

PROCEDURES PERFORMED

HEPATIC HYDATID

TABLE NO 8
Total No. of Cases 21

POCEDURE	TOTAL	PERCENTAGE
CONCERVATIVE	1	4.76%
PAIR	2	9.52%
PARTIAL CYST EXCISION & EVACUATION & DRAINAGE	10	47.6%
EXCITION& CAPITONAGE	1	4.76%
EXCISION&OMENTOPLASTY	3	14.28%
EXCITION & OMENTOPLASTY & CHOLDOCHOTOMY & T” TUBE DRAINAGE	1	4.76%
PERICYSTECTOMY	1	4.76%
LIVER RESECTION	0	0%
LIVER TRANSPLANTATION	0	0%
MARSUPULISATION	0	0%

SPLENIC HYDATID

TABLE NO 9

Total No. of Cases 2

PROCEDURE	NO OF CASES	PERCENTAGE
SPLENECTOMY	2	9.52%
MARSUPULISATION	0	0%

In this series both the cases were having a huge cyst entirely replacing spleen. So splenectomy was done in all the cases.

RENAL HYDATID

TABLE NO 10

No. of Cases 1

PROCEDURE	NO OF CASES	PERCENTAGE
WEDGE RESECTION	1	4.76%
EXISION OF CYST	0	0%

As the cyst is deeply situated in the cortex wedge resection offers 100% cure.

RETRO PERITONEAL HYDATID CYST

TABLE NO 11

No. of Cases 1

PROCEDURE	NO OF CASES	PERCENTAGE
EXCISION	1	4.76%

PERITONEAL HYDATID CYST

TABLE NO 12

No. of Cases 1

PROCEDURE	NO OF CASES	PERCENTAGE
EXCISION	1	4.76%

Peritoneal involvement in this series was secondary to liver involvement. Thorough peritoneal wash with hyper tonic saline was given along with excision of cyst in liver and peritoneum.

DISCUSSION

According to various studies of the hydatid disease it is clearly proved that the majority of cases show hydatid in the liver (first filter) by spleen and then only other abdominal organs like Kidney, Peritoneum, etc., In this series, males are affected slightly more than the females. ESR is elevated only in 35% of cases. There is also a generalized. Eosinophilia in 85% of cases in this series. Hydatid cysts can occur in any age group. In other series disease is not recorded below 3 years of age. In this series the minimum age was 23 years maximum age was 65 years and the average age was 39.5 years. In this series all the patients had definite contact with pet dogs.

Allergic manifestations were not present in any case in this study. Only one case presented with biliary communication and Jaundice. Only 4 patients were asymptomatic in this series. Investigations like casonis test and other serological tests were not available for use in this series. X-ray USG and CT scan can be very useful diagnostic tools. Right side of liver is affected more than the left. In this series 50% of hydatid liver cysts were in the right. Both the lobes in 39% of cases and left lobe is affected in 11% of cases. In hydatid cysts in other sites, the presence of cystic swelling is an important clinical picture. The valve of hydatid thrill as a diagnostic sign is doubtful. This sign is unmistakable if it is present.

The hydatid cyst in other places may sometime give rise to diagnostic difficulties and only a differential diagnosis can be offered. After the advent of ultrasound and CT scan the diagnosis is made easier. Since the prevalence of hydatid disease in and around Madurai is common. It may also be included in the differential diagnosis of a cystic swelling situated anywhere in the body.

Regarding treatment it is agreed that if there are no other contraindications surgery offers the only reasonable treatment and a safeguard for the future against the very real hazard of growth of the cyst and other complications. In the present series partial excision of cyst evacuation and external drainage is done for 47.6% of cases. Percutaneous aspiration injection, re-aspiration was done in 9.52% of cases, Excision & evacuation with omentoplasty was done in 14.28% of cases; 4.76% of cases (1 patient) managed conservatively. Cholecystotomy with 'T' tube drainage was done in 1 case (4.76%). Splenectomy was done in 9.52% of cases. Wedge resection of renal hydatid was done in 1 case. Liver resection, liver transplantation, marsupialisation was not done in this series. Albendazole was given preoperatively for all patients. It may be seen from the present data that active transmission of hydatid disease occurs in and around Madurai and the main species of parasite involved is *Echinococcus granulosus* and cattle are the main animal reservoir of infestation.

CONCLUSION

A general account of the parasite has been discussed. The incidence of human hydatid infection in Govt. Rajaji Hospital has been reviewed. The numbers of cases in relation to the specific organs are given in detail along with statistical data. The choices of operations were discussed. Since hydatid disease is prevalent in Madurai and its suburbs it can be considered as an endemic area for hydatid disease. In this study male members were affected more and the most common site of infestation was the liver. After the advent of Chemotherapy it has been used as an adjuvant treatment for preoperative period and recurrence and also for inoperable disease. Even though the incidence of hydatid disease is less the disease is still active and therefore it is better to take prophylactic measures to prevent the disease from spreading by making the people aware of the mode of spread and their complications.

BIBLIOGRAPHY

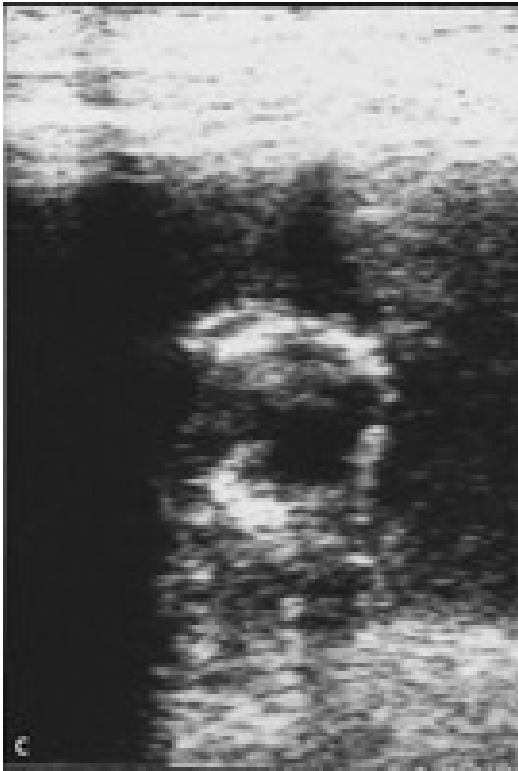
1. Abdul Salaam M. (1968). Research needs in Echinococcosis. W.H.O. vol 39 p101-113.
2. Barrett N.R (1949). Lancet 2 p234.
3. Chatterjee K.D (1952) Human parasites and parasitic diseases.
4. Dew H.R (1926) Mechanism of Daughter Cyst Formation in Hydatid Disease. J Aug 01 p451-460.
5. Dew H.R (1967) Clinical Parasitology VI edition
6. Gamell M.A (1968) Control measures against Hydatid Disease W.H.O vol.30 p73-100.
7. Gupta (1966) JIMA vol.12 p49
8. Ian Aird (1958) Companion to Surgical Studies.
9. Kagan I.G. (1968) Serological tests for the diagnosis of Hydatid Disease
10. Lakshmanan D. (1965) The antiseptic vol.62 p947.
11. Rao A.R and Balasubramanian V. (1957) JIS vol.19 p42.
12. Rausch R.L (1968) Taxonomic characters in Genus Echinococcus W.H.O. vol.39 p1-4.
13. Reddy and Anguli (1953) JIS vol.15 p100.

14. Sadanandam A.D (1969) Studies on Echinococcus for the degree of M.Phil. M.K. University.
15. Smith J.D (1968) Host Specificity of Echinococcus W.H.O vol.39 p5-12
16. Thatcher V.E. & Sousa O.E (1966) Annual Tropical Medical Parasite vol.60 p405-416.
17. Vasudevan (1967) A Clinical Study of Hydatid Disease in Madurai, Madras University.
18. Manson's Tropical Medicine.
19. Sabiston Text Book of Surgery p1087-1089.
20. Blumgart Biliary Hydatid disease p247-249.
21. Recent Advances in Surgery.

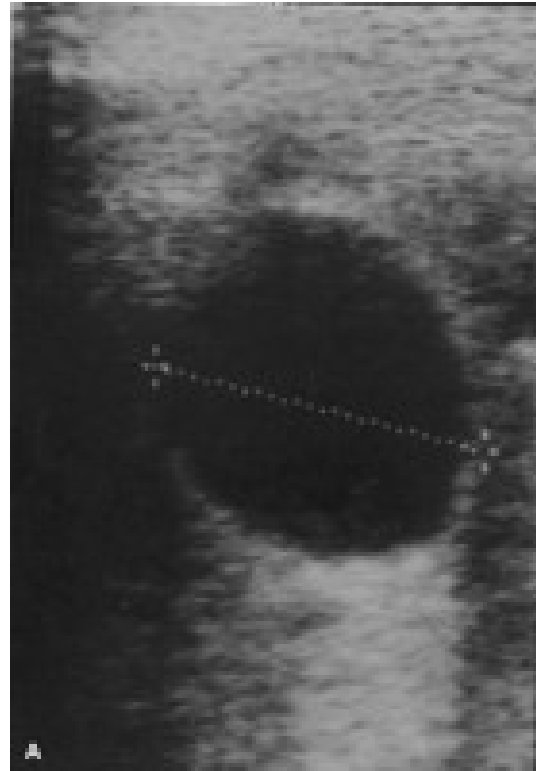
DETAILS OF THE PATIENTS EXAMINED

S.No	NAME	AGE / SEX	IP.NO	DATE OF ADMISSION	DIAGNOSIS	TREATMENT	BLOOD GROUP	ESR
1	Saraswathy	60/f	75109	6/5/2008	Rt lobe cyst	Pericystectomy	O+ve	40mm
2	Samayan	40/m	69847	4/2/2008	Rt lobe cyst & cbd obstruction	Excision & omentoplasty & CBD Exploration & T' Tube drainage	B+ve	42mm
3.	Saravanakumar	29/m	85039	7/8/2008	Left lobe cyst	PAIR	A+ve	5mm
4.	Panchavarnam	27/f	62465	31/7/2008	Rt lobe cyst	Partial excision & drainage	O-ve	3mm
5.	panthanam	35/f	34439	10/4/2008	Multiple liver cyst	Partial excision & drainage	O+ve	6mm
6.	Meenakshi	32/f	52823	28/7/2007	Rt lobe cyst	PAIR	B+ve	4mm
7.	Nirmala	39/f	87372	21/11/2007	Multiple liver cyst	Excision & capitonage	B+ve	35mm
8.	Saroja	45/f	97749	11/1/2008	Rt lobe cyst	Partial excision & drainage	B+ve	3mm
9.	Senthil kumar	23/m	29578	7/03/2008	Rt lobe cyst	Partial excision & drainage	B+ve	5mm
10.	Marimuthu	40/m	51895	20/7/2007	Retroperitoneal cyst	Excision of cyst	O+ve	30mm
11.	Thiruppathy	65/m	23200	18/1/2007	Splenic cyst	Splenectomy	A+ve	25mm

12.	Karmegam	32/m	65674	1/08/06	Rt lobe cyst	Partial excision & drainage	B+ve	3mm
13.	Shanmugaraj	52/m	69451	13/9/06	Left lobe cyst	Partial excision & drainage	O+ve	6mm
14.	Kamatchi	50/f	77987	19/11/07	Multiple liver cyst & peritoneal cyst	Partial excision & drainage	B+ve	60mm
15.	Sarasu	60/f	73380	22/09/2008	Multiple liver cyst	Partial excision & drainage	O+ve	14mm
16.	Christy	54/f	30641	18/10/2008	Rt lobe cyst	Excision & omentoplasty	B+ve	3mm
17.	Lakshmanan	27/m	84402	9/10/2008	Calcified small trope cyst	Conservative management	O-ve	2mm
18.	Ponnammal	49/f	36543	4/5/2007	Multiple liver cyst & rt renal cyst	Partial excision & drainage & wedge resection for renal cyst	O+ve	68mm
19.	Sivaraman	32/m	56743	3/8/2007	Multiple liver cyst	Excision & omentoplasty	O+ve	4mm
20.	Santhanam	37/m	98070	6/12/2007	Multiple liver cyst	Partial excision & drainage	O+ve	6mm
21.	Nithya	18/f	67522	4/3/2008	Splenic cyst	Splenectomy	AB-ve	8mm



Calcified cyst



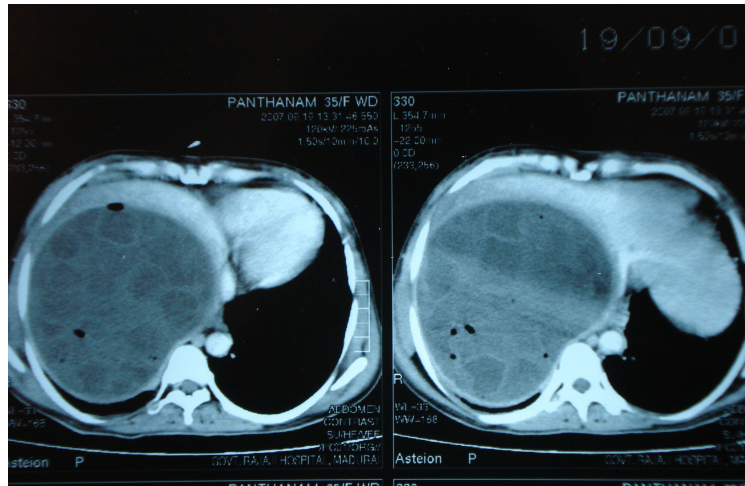
Uni locular hydatid cyst liver



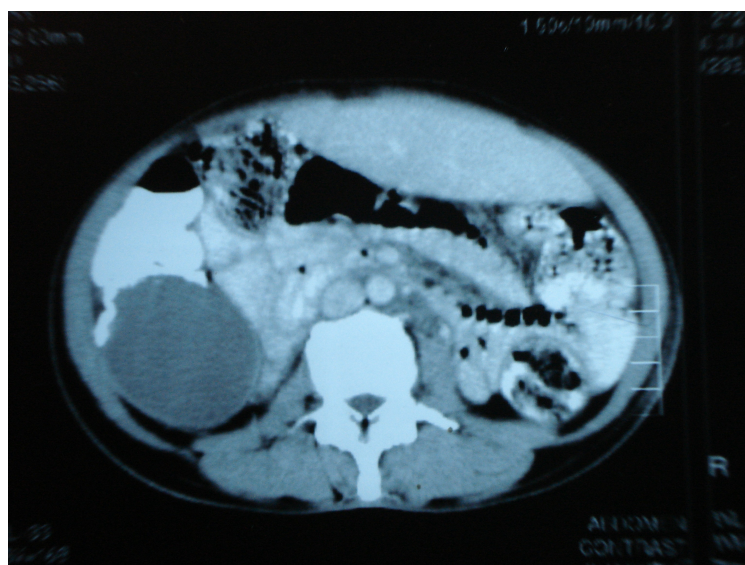
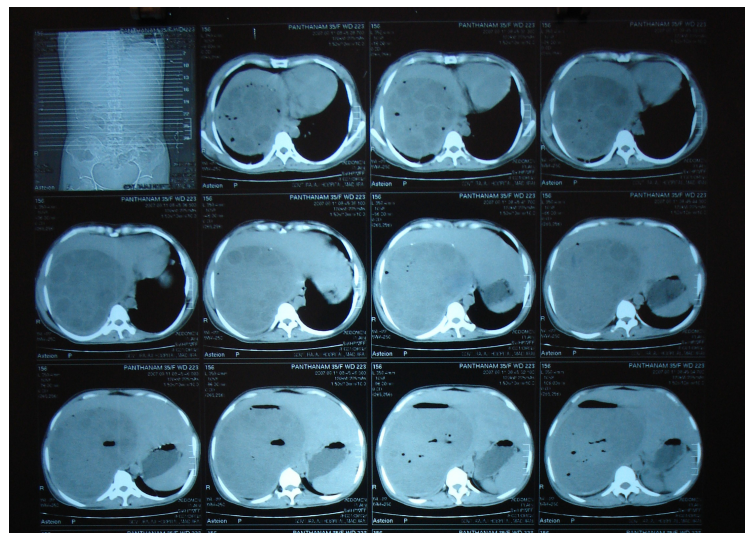
Daughter cysts



Water lily sign

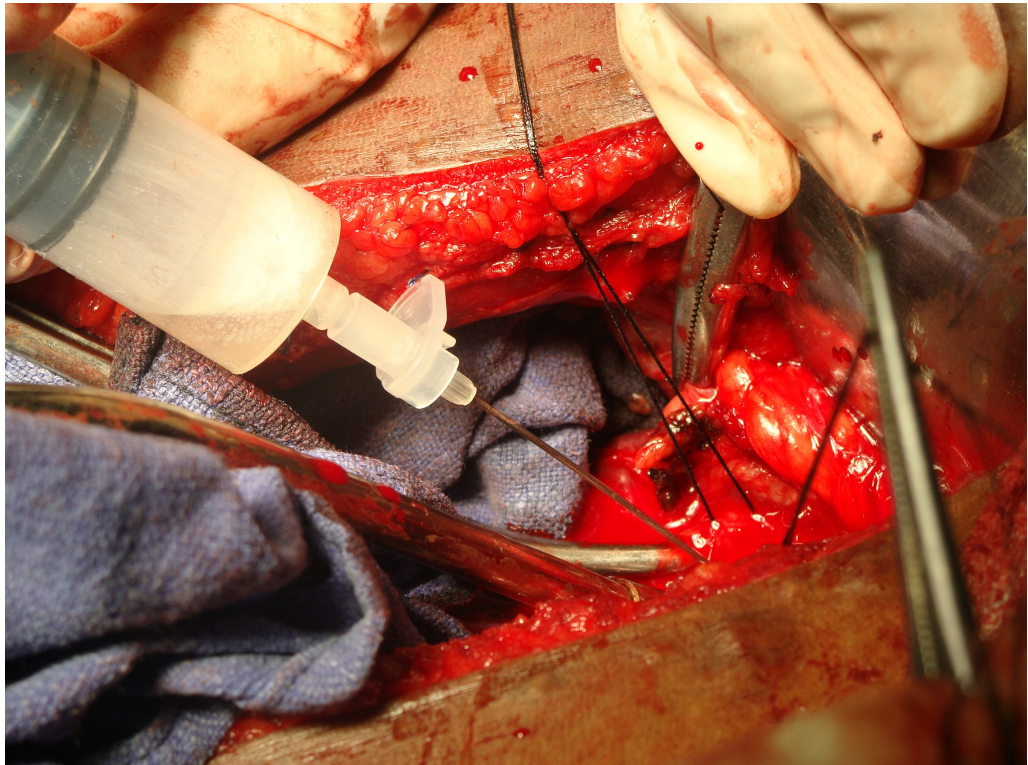


CT of multiple liver hydatid

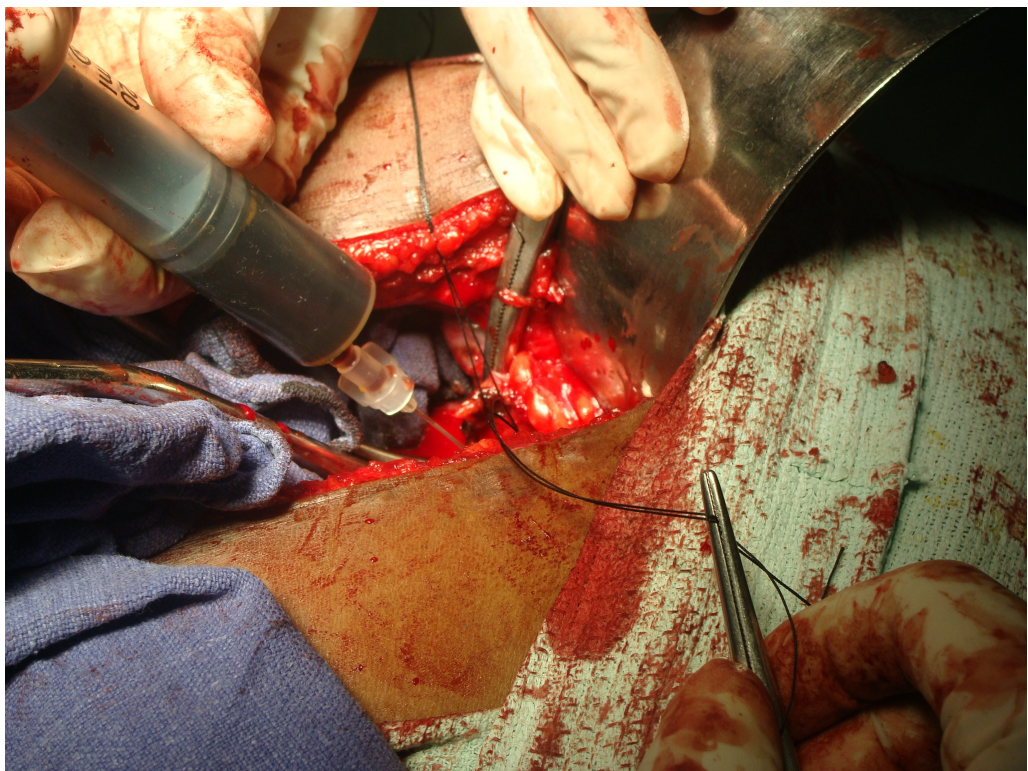


Right renal hydatid

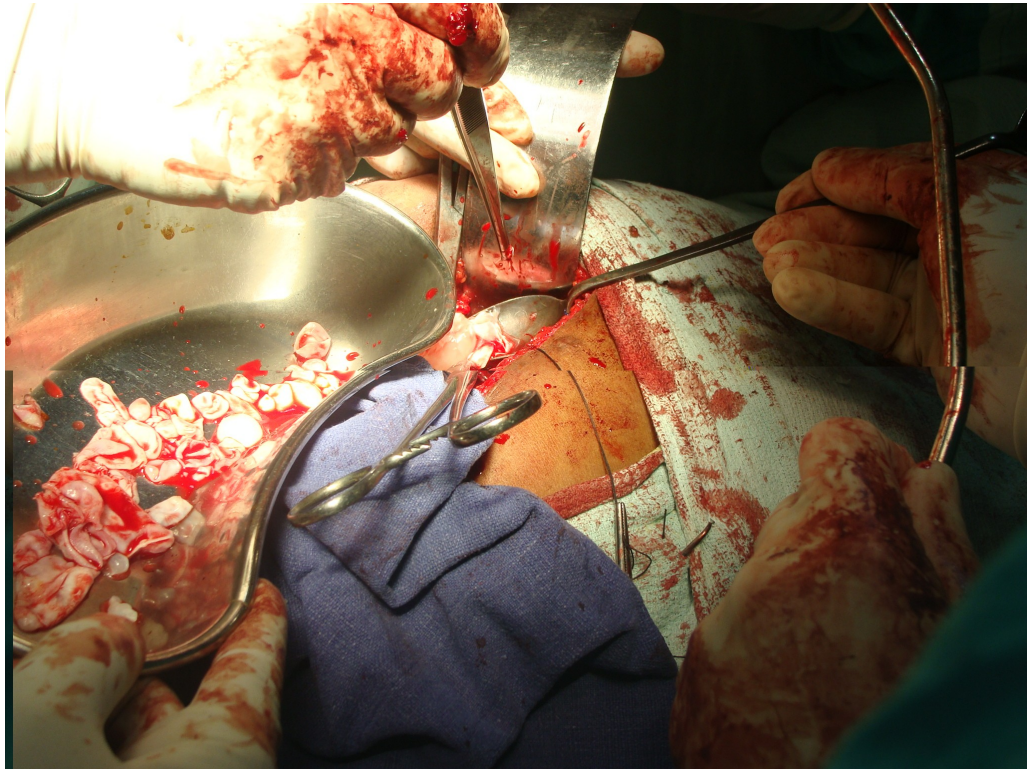
CYST EVACUATION & EXTERNAL DRAINAGE



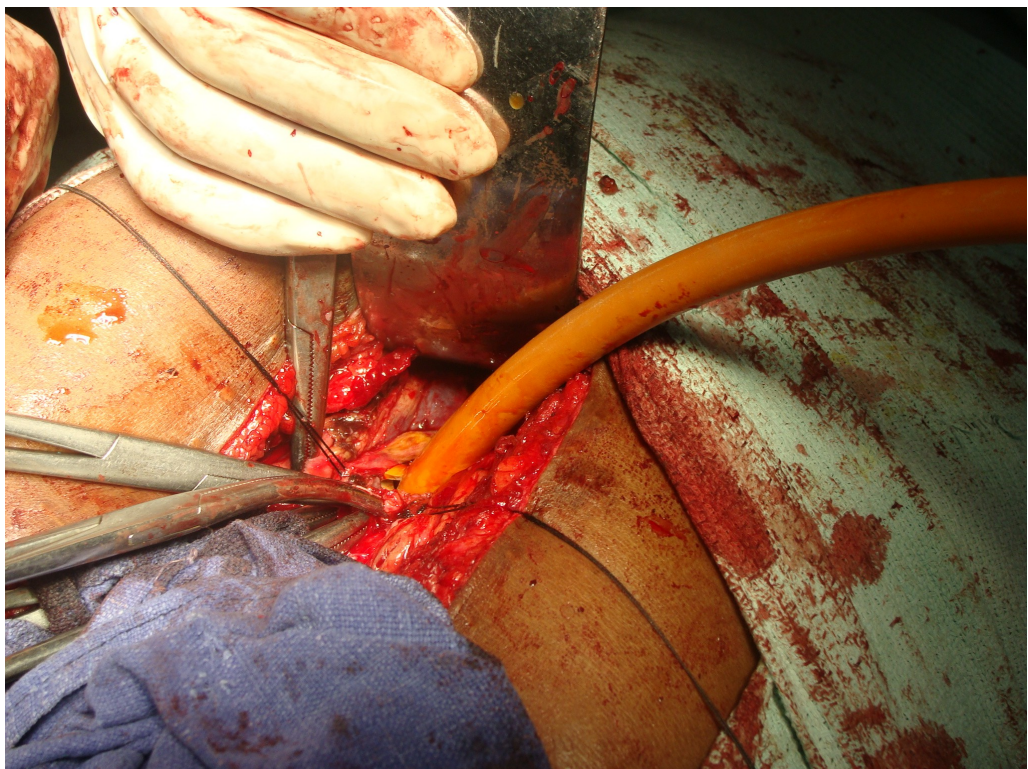
Aspiration of cystic fluid



Injection of scolicide



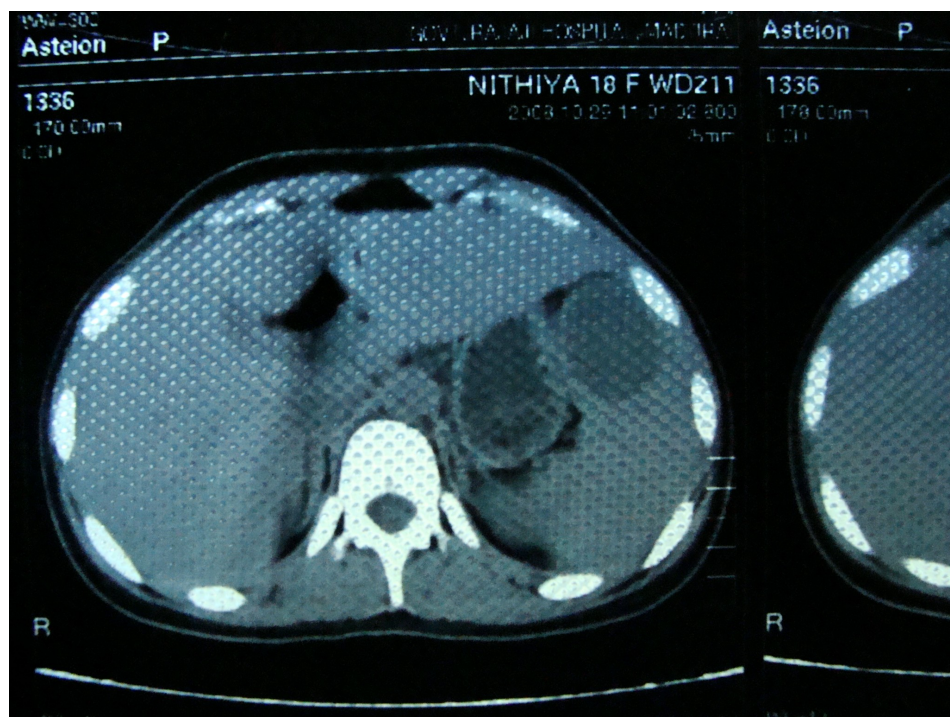
Scooping of daughter cysts



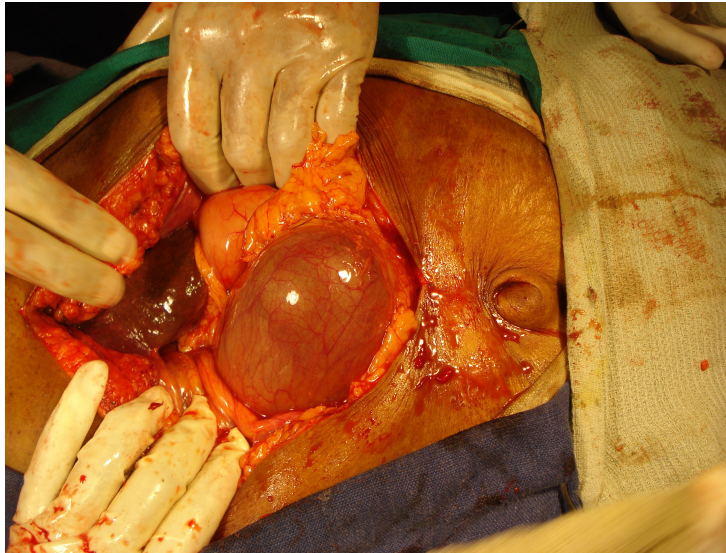
External mallecots drainage



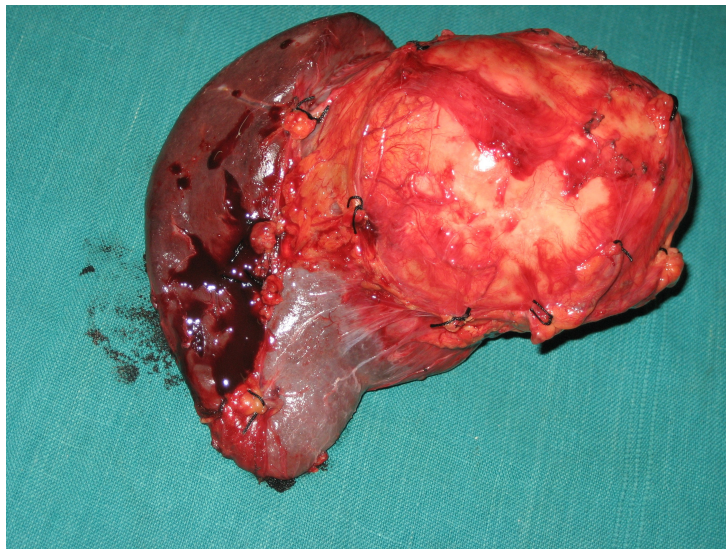
USG of splenic hydatid



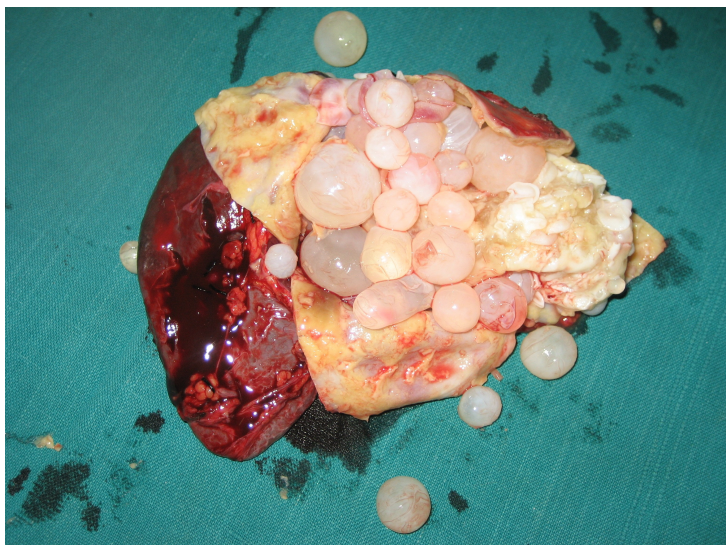
CT of splenic hydatid



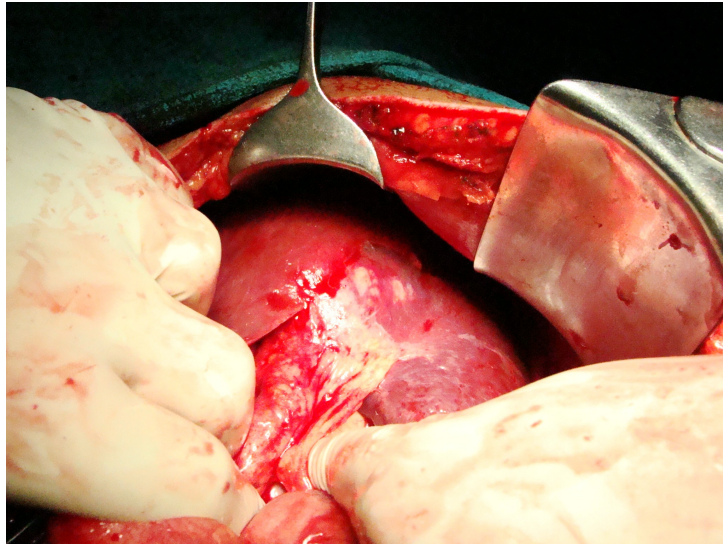
Hydatid spleen during exploration



Splenic hydatid after removal



Hydatid spleen with daughter cysts



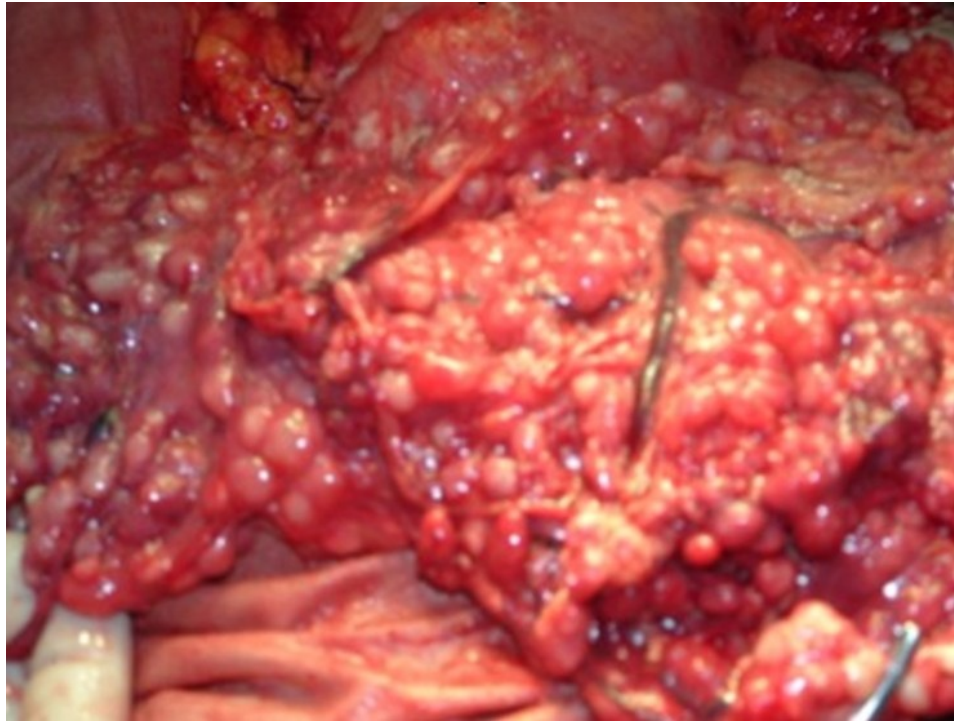
Splenic hydatid during exploration



Spleen delivered through after betadine towel package



Splenectomy specimen showing cyst



Hydatidosis-of omentum



Hydatidosis-retro peritoneal